```
=> s flap endonuclease
L1          865 FLAP ENDONUCLEASE

=> d rem l1
'REM' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):dup rem l1
'DUP' IS NOT A VALID FORMAT
'REM' IS NOT A VALID FORMAT
'L7' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2           305 DUP REM L1 (560 DUPLICATES REMOVED)

=>

=>

=>

=>

=>

=>

=> s l2 and (dna or rna or polynucleotide or double flap)
L3          294 L2 AND (DNA OR RNA OR POLYNUCLEOTIDE OR DOUBLE FLAP)

=> focus l3
PROCESSING COMPLETED FOR L3
L4           294 FOCUS L3 1-

=> d l4 1-15 ibib ab

L4    ANSWER 1 OF 294  BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
ACCESSION NUMBER: 2003-14605  BIOTECHDS
TITLE:            A composition for introducing two or more mutations to a
                  target DNA molecule in an amplification reaction,
                  useful for multi-site directed mutagenesis, comprises a
                  DNA polymerase, a DNA ligase and a
                  flap endonuclease;
                     involving vector-mediated restriction endonuclease gene
                     transfer and expression in Escherichia coli for use in
                     site-directed mutagenesis and DNA shuffling
AUTHOR:           CLINE J M; HOGREFE H H
PATENT ASSIGNEE:  STRATAGENE
PATENT INFO:      WO 2003025118 27 Mar 2003
APPLICATION INFO: WO 2002-US22759 18 Jul 2002
PRIORITY INFO:    US 2001-307927 26 Jul 2001; US 2001-307927 26 Jul 2001
DOCUMENT TYPE:    Patent
LANGUAGE:         English
OTHER SOURCE:     WPI: 2003-354591 [33]
AB    DERWENT ABSTRACT:
      NOVELTY - Composition for introducing two or more mutations to a target
      DNA molecule in an amplification reaction comprising a
```

DNA polymerase, a **DNA** ligase and a **flap endonuclease**, also optionally comprising a selection enzyme with or without a host cell for transformation, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following: (1) kits for introducing two or more mutations to a target **DNA** molecule in an amplification reaction comprising the composition cited above and packaging means; (2) introducing two or more mutations to a target **DNA** molecule; and (3) **DNA** shuffling method.

BIOTECHNOLOGY - Preferred Composition: The composition further comprises ATP and NAD having a concentration of 0.02-0.2 mM, preferably 0.1 mM, per reaction. The **DNA** polymerase is a thermostable **DNA** polymerase, such as Taq, Pfu, Tma, Tli, KOD, JDF-3, PGB-D, Tgo, or Pyrolobus furmarius **DNA** polymerase. Preferably, the Pfu **DNA** polymerase is Pfu-Turbo **DNA** polymerase. The **DNA** ligase is a thermostable **DNA** ligase, such as Pfu, Tth, Taq, Thermus filiformis, Rhodothermus marinus, Thermus scotoductus, or Bacillus stearothermophilus **DNA** ligase. The **flap endonuclease** is a thermostable **flap endonuclease**, such as FEN-1, RecJ or Dna2. Preferably, the composition comprises Pfu **DNA** polymerase with a concentration of 1.25-2.5 U per 25 microl reaction, Taq **DNA** ligase with a concentration of 10-20 U per 25 microl reaction, and FEN-1 with a concentration of 400 ng-4 microg per 25 microl reaction. The composition further comprises 0.01-0.2mM NAD per reaction. The composition preferably comprises 2.5 U Pfu **DNA** polymerase, 15 U Taq **DNA** ligase, 400 ng per 25 microl reaction FEN-1, and 0.1 mM NAD. The selection enzyme is a restriction endonuclease, which is methylation-dependent. The methylation-dependent restriction endonuclease is DpnI, Nan II, NmuD I, or NmuE I. The composition further comprises a polymerase enhancing factor and at least one primer, preferably a degenerate primer. The composition comprising a host cell for transformation further comprises DMSO. The host cell is an Escherichia coli cell. Preferred Method: Introducing two or more mutations to a target **DNA** molecule comprises annealing one or more primers to the same strand of the **DNA** molecule, where each primer comprises at least one mutation site with respect to the **DNA** molecule, synthesizing by means of an amplification reaction a mutagenized single strand of **DNA** comprising the primers in the presence of a **DNA** polymerase, a **DNA** ligase and a **flap endonuclease**, and digesting the non-mutagenized strands of the **DNA** molecule with a selection enzyme to produce a **DNA** product. Alternatively, the method comprises annealing one or more primers to the same strand of the **DNA** molecule, where each primer comprises at least one mutation site with respect to the **DNA** molecule, synthesizing by means of an amplification reaction a mutagenized single strand **DNA** comprising the primers in the presence of a **DNA** polymerase, a **DNA** ligase and a **flap endonuclease**, digesting the non-mutagenized strands of the **DNA** molecule with a selection enzyme to produce a **DNA** product, and transforming a host cell with the **DNA** product. The method may also comprise annealing one or more primers to the same strand of the **DNA** molecule, where each primer comprises at least one mutation site with respect to the **DNA** molecule, synthesizing by means of an amplification reaction a mutagenized single strand **DNA** comprising the primers in the presence of a **DNA** polymerase, a **DNA** ligase and a **flap endonuclease**, digesting the non-mutagenized strands of the **DNA** molecule with a selection enzyme, generating a double-stranded mutagenized **DNA** intermediate, and transforming a host cell with the double-stranded mutagenized **DNA** intermediate. **DNA** shuffling comprises fragmenting one or more target polynucleotides, preferably double-stranded target polynucleotides, into **polynucleotide** fragments, providing the **polynucleotide** fragments in an amplification reaction in the

presence of a **DNA** polymerase, a **polynucleotide** template, a **DNA** ligase and a **flap endonuclease** to produce an amplified product, and transforming a host cell with the product from the amplification reaction. The endonuclease is a **flap endonuclease**, a **DNA** polymerase deficient in 5'-3' exonuclease activity, or a **DNA** polymerase deficient in **DNA** polymerase activity. The **DNA** polymerase deficient 5'-3' exonuclease activity or a **DNA** polymerase deficient in **DNA** polymerase activity is Taq **DNA** polymerase, Tth **DNA** polymerase, or Tma **DNA** polymerase. The method further comprises selecting a subpopulation of the **polynucleotide** fragments after fragmenting the target polynucleotides. The amplification reaction in any of the methods above further comprises a polymerase enhancing factor, ATP and NAD having a concentration of 0.02-0.2 mM per reaction. Amplification is performed in the presence of DMSO. The amplification reaction comprises 3-60 reaction cycles. The target **DNA** molecule is a circular plasmid **DNA**. The same strand is a first strand of a double-stranded target **DNA** molecule. The methods further comprise annealing one or more primers to a second strand of the double-stranded target **DNA** molecule. Each primer used in any of the methods comprises a different mutation site with respect to the **DNA** molecule.

USE - The compositions and methods are useful for introducing two or more mutations to a target **DNA** molecule, particularly for multi-site directed mutagenesis. The method is also useful for **DNA** shuffling. The kits are useful for performing multi-site directed mutagenesis and **DNA** shuffling.

ADVANTAGE - The present compositions and methods provide high mutation frequencies and increased number of transformants compared to prior art. High mutation efficiency and large number of transformants allow one to sequence only a few clones in order to identify the correct mutants and to obtain the desired mutant by screening large number of transformants in a short time. (83 pages)

L4 ANSWER 2 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:960111 HCAPLUS
DOCUMENT NUMBER: 138:315616
TITLE: Interaction of Replication Protein A and **Flap Endonuclease** 1 with **DNA** Duplexes Containing a Nick or a Flap
AUTHOR(S): Khlimankov, D. Yu.; Rechkunova, N. I.; Khodyreva, S. N.; Petruseva, I. O.; Nazarkina, Zh. K.; Belousova, E. A.; Lavrik, O. I.
CORPORATE SOURCE: Siberian Division, Novosibirsk Institute of Bioorganic Chemistry, Russian Academy of Sciences, Novosibirsk, 630090, Russia
SOURCE: Molecular Biology (Moscow, Russian Federation, English Edition) (Translation of Molekulyarnaya Biologiya) (2002), 36(6), 849-856
CODEN: MOLBBJ; ISSN: 0026-8933
PUBLISHER: MAIK Nauka/Interperiodica Publishing
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nicks and flaps are intermediates in various processes of **DNA** metab., including replication and repair. Photoaffinity modification was employed in studying the interaction of the replication protein A (RPA) and **flap endonuclease** 1 (FEN-1) with **DNA** duplexes similar to structures arising during long-patch base excision repair. The proteins were also tested for effect on **DNA** polymerase .beta. (Pol.beta.) interaction with **DNA**. Using Pol.beta., a photoreactive dTTP analog was added to the 3 end of an oligonucleotide flanking a nick or a flap in **DNA** intermediates. The character and intensity of protein labeling depended on the type of intermediates and on the presence of the phosphate or THF at the 5 end of

a nick or a flap. Photoaffinity labeling of Pol.beta. substantially (up to three times) increased in the presence of RPA or FEN-1. Various **DNA** substrates were used to study the effects of RPA and FEN-1 on Pol.beta.-mediated **DNA** synthesis with displacement of a downstream primer. In contrast to FEN-1, RPA had no effect on **DNA** repair synthesis by Pol.beta. during long-patch base excision repair.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:242113 HCAPLUS
DOCUMENT NUMBER: 138:249748
TITLE: Use of gap-sensitive **flap endonuclease** cleavage in detection of single nucleotide polymorphisms
INVENTOR(S): Meng, Fan; Watson, Stanley J.; Akil, Huda; Taylor, Larry P.
PATENT ASSIGNEE(S): The Regents of the University of Michigan, USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003024314	A2	20030327	WO 2002-US29869	20020919
WO 2003024314	A3	20030814		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-323466P P 20010919

AB Methods and compns. for detecting a single nucleotide, in particular, a single nucleotide polymorphism (SNP) in a target nucleic acid mol. using a gap-sensitive **flap endonuclease** are disclosed. The methods involve annealing upstream and downstream probes to a target nucleic acid mol. such that an overlapping, non-complementary portion of the downstream probe forms a closed flap structure at the SNP site if it is present. If the SNP is not present an open flap structure forms with a gap at the SNP site. The closed flap structure is cleaved by a gap-sensitive **flap endonuclease** enzyme indicating the SNP is present in the target nucleic acid. Cleavage is indicated by release of a reporter group, e.g. by liberation of one member of a FRET pair resulting in a fluorescence signal. The method can be adapted for use in microarrays and several different methods of using microarrays are described.

L4 ANSWER 4 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:199548 HCAPLUS
DOCUMENT NUMBER: 130:278601
TITLE: Cloning of gene for highly thermostable **Flap endonuclease** of Pyrococcus horikoshii
INVENTOR(S): Matsui, Ikuo; Ishikawa, Kazuhiko; Kosugi, Yoshiji; Matsui, Eriko; Kawasaki, Satoko
PATENT ASSIGNEE(S): Agency of Industrial Sciences and Technology, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 8 pp.

CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 11075849	A2	19990323	JP 1997-239440	19970904
JP 3018163	B2	20000313		
US 6251649	B1	20010626	US 1998-146319	19980903
US 6255081	B1	20010703	US 1998-175973	19981021

PRIORITY APPLN. INFO.: JP 1997-239440 A 19970904
US 1998-146319 A3 19980903

AB The gene for a highly thermostable **Flap endonuclease** is isolated from Pyrococcus horikoshii strain JCM9974 and its encoded amino acid sequence (343 residues) deduced. The enzyme exhibits a pH optimum 6.0-8.0 and temp. optimum >75.degree.. It remains 50% active after heating at 95.degree. for 5 h. It is denatured at 103.degree.. Methods for recombinant prepn. of the enzyme are claimed.

L4 ANSWER 5 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:505792 HCAPLUS
DOCUMENT NUMBER: 129:227284
TITLE: Flexible loops and helical arches
AUTHOR(S): Sayers, Jon R.; Artymiuk, Peter J.
CORPORATE SOURCE: Krebs Institute, Division of Molecular and Genetic Medicine, University of Sheffield, Sheffield, S10 2JF, UK
SOURCE: Nature Structural Biology (1998), 5(8), 668-670
CODEN: NSBIEW; ISSN: 1072-8368
PUBLISHER: Nature America
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 12 refs. The crystal structure of an archaeal 5' **flap endonuclease** suggests a common mode of action in this divergent group of essential replicative enzymes. The discussion is based mainly on the structure of Methanococcus jannaschii 5'-nuclease [MjFEN-1 (**flap endonuclease** 1)] which consists of a large flexible loop mounted on a globular domain contg. the active site.

REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:156917 HCAPLUS
DOCUMENT NUMBER: 137:164493
TITLE: The roles of Klenow processing and flap processing activities of **DNA** polymerase I in chromosome instability in Escherichia coli K12 strains
AUTHOR(S): Nagata, Yuki; Mashimo, Kazumi; Kawata, Masakado; Yamamoto, Kazuo
CORPORATE SOURCE: Department of Biomolecular Sciences, Tohoku University, Sendai, 980-8578, Japan
SOURCE: Genetics (2002), 160(1), 13-23
CODEN: GENTAE; ISSN: 0016-6731
PUBLISHER: Genetics Society of America
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The sequences of spontaneous mutations occurring in the endogenous tonB gene of Escherichia coli in the .DELTA.polA and polA107 mutant strains were compared. Five categories of mutations were found: (1) deletions, (2) minus frameshifts, (3) plus frameshifts, (4) duplications, and (5) other mutations. The .DELTA.polA strain, which is deficient in both Klenow domain and 5' .fwdarw. 3' exonuclease domain of **DNA** polymerase I, shows a marked increase in categories 1-4. The polA107

strain, which is deficient in the 5' .fwdarw. 3' exonuclease domain but proficient in the Klenow domain, shows marked increases in categories 3 and 4 but not in 1 or 2. Previously, the authors reported that the polA1 strain, which is known to be deficient in the Klenow domain but proficient in the 5' .fwdarw. 3' exonuclease domain, shows increases in categories 1 and 2 but not in 3 or 4. The 5' .fwdarw. 3' exonuclease domain of **DNA** polymerase 1 is a homolog of the mammalian FEN1 and the yeast RAD27 flap nucleases. The authors therefore proposed the model that the Klenow domain can process deletion and minus frameshift mismatch in the nascent **DNA** and that flap nuclease can process plus frameshift and duplication mismatch in the nascent **DNA**.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:633170 HCAPLUS
DOCUMENT NUMBER: 139:174802
TITLE: **RNA** target detection using INVADER oligonucleotide-directed cleavage using thermostable derivatives of **DNA** polymerases with thermostable 5'-nuclease activities
INVENTOR(S): Lyamichev, Victor; Neri, Bruce P.; Hall, Jeff; Lukowiak, Andrew A.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 392 pp., Cont.-in-part of U. S. Ser. No. 713,601.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003152971	A1	20030814	US 2002-290386	20021107
US 5985557	A	19991116	US 1996-756386	19961126
WO 9727214	A1	19970731	WO 1997-US1072	19970122
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
WO 9842873	A1	19981001	WO 1998-US5809	19980324
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6348314	B1	20020219	US 1999-350309	19990709
US 2004072182	A1	20040415	US 2003-356861	20030203

PRIORITY APPLN. INFO.:

US 1996-756386	A3	19961126
WO 1997-US1072	A	19970122
WO 1998-US5809	A	19980324
US 1999-350309	A2	19990709
US 2000-713601	A2	20001115
US 2001-344946P	P	20011107
US 2002-361060P	P	20020227
US 1996-599491	A2	19960124
US 1996-682853	A2	19960712
US 1996-758314	A	19961202
US 1996-759038	A	19961202
US 1997-823516	A	19970324
US 2002-290386	A2	20021107

AB The present invention relates to compns. and methods for the detection and characterization of nucleic acid sequences and variations in nucleic acid sequences. The present invention relates to methods for forming a nucleic acid cleavage structure on a target sequence and cleaving the nucleic acid cleavage structure in a site-specific manner. For example, in some embodiments, a 5' nuclease activity from any of a variety of enzymes is used to cleave the target-dependent cleavage structure, thereby indicating the presence of specific nucleic acid sequences or specific variations

thereof. The present invention provides novel cleavage agents and polymerases for the cleavage and modification of nucleic acid. The cleavage agents and polymerases find use, for example, for the detection and characterization of nucleic acid sequences and variations in nucleic acid sequences. In some embodiments, the 5'-nuclease activity of a variety of modified Thermus polymerase enzymes is used to cleave a target-dependent cleavage structure, thereby indicating the presence of specific nucleic acid sequences or specific variations thereof. The term "cleavage structure" refers to a structure that is formed by the interaction of at least one probe oligonucleotide (called the INVADER oligonucleotide) and a target nucleic acid, forming a structure comprising a duplex, the resulting structure being cleavable by a cleavage agent including but not limited to an enzyme. A sample suspected of contg. the target sequence is contacted with oligonucleotides capable of forming an invasive cleavage structure in the present of the target sequence and with an agent for detecting the presence of the invasive cleavage structure. ARRESTOR oligonucleotides improve sensitivity of multiple sequential invasive cleavage assays and allow use of higher concns. of primary probe without increasing background signal. The detailed description of the invention includes: (1) improved enzymes for the use in INVADER oligonucleotide-directed cleavage reactions comprising **RNA** targets; (2) reaction design for INVADER assay detection of **RNA** targets; (3) kits for performing the **RNA** invader assay; and (4) the INVADER assay for direct detection and measurement of specific **RNA** analytes.

L4 ANSWER 8 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:	1999:195723 HCAPLUS
DOCUMENT NUMBER:	131:2203
TITLE:	Processing of UV Damage in Vitro by FEN-1 Proteins as Part of an Alternative **DNA** Excision Repair Pathway
AUTHOR(S):	Yoon, Jung-Hoon; Swiderski, Piotr M.; Kaplan, Bruce E.; Takao, Masashi; Yasui, Akira; Shen, Binghui; Pfeifer, Gerd P.
CORPORATE SOURCE:	Department of Biology Department of Molecular Biology and Department of Cell and Tumor Biology, Beckman Research Institute of the City of Hope, Duarte, CA, 91010, USA
SOURCE:	Biochemistry (1999), 38(15), 4809-4817 CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER:	American Chemical Society
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB UV irradn. induces predominantly cyclobutane and (6-4) pyrimidine dimer photoproducts in **DNA**. Several mechanisms for repairing these mutagenic UV-induced **DNA** lesions have been identified. Nucleotide excision repair is a major pathway, but mechanisms involving photolyases and **DNA** glycosylases have also been characterized. Recently, a novel UV damage endonuclease (UVDE) was identified that initiates an excision repair pathway different from previously established repair mechanisms. Homologues of UVDE have been found in eukaryotes as well as in bacteria. In this report, we have used oligonucleotide substrates contg. site-specific cyclobutane pyrimidine dimers and (6-4) photoproducts for the characterization of this UV damage repair pathway. After introduction of single-strand breaks at the 5' sides of the photolesions by UVDE, these intermediates became substrates for cleavage by flap endonucleases (FEN-1 proteins). FEN-1 homologues from humans, Saccharomyces cerevisiae, and Schizosaccharomyces pombe all cleaved the UVDE-nicked substrates at similar positions 3' to the photolesions. T4 endonuclease V-incised **DNA** was processed in the same way. Both nicked and flapped **DNA** substrates with photolesions (the latter may be intermediates in **DNA** polymerase-catalyzed strand displacement synthesis) were cleaved by FEN-1. The data suggest that the two enzymic activities, UVDE and FEN-1, are part of an alternative

excision repair pathway for repair of UV photoproducts.
REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 9 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:431766 HCAPLUS
DOCUMENT NUMBER: 138:53396
TITLE: Regulation of **DNA** replication fork genes by 17.beta.-estradiol
AUTHOR(S): Lobenhofer, Edward K.; Bennett, Lee; Cable, P. Louann; Li, Leping; Bushel, Pierre R.; Afshari, Cynthia A.
CORPORATE SOURCE: Gene Regulation Group, National Institute of Environmental Health Sciences, Research Triangle Park, NC, 27709, USA
SOURCE: Molecular Endocrinology (2002), 16(6), 1215-1229
CODEN: MOENEN; ISSN: 0888-8809
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The steroid hormone estrogen can stimulate mitogenesis in hormone-responsive breast cancer epithelial cells. This action is attributed to the transcriptional activity of the ER, a ligand-dependent transcription factor. However, the exact mol. mechanism underlying estrogen-induced proliferation has yet to be completely elucidated. Using custom cDNA microarrays contg. many genes implicated in cell cycle progression and **DNA** replication, we examd. the gene expression of a hormone-responsive breast cancer cell line (MCF-7) treated with a mitogenic dose of estrogen in the absence of confounding growth factors found in serum. Gene expression changes were monitored 1, 4, 12, 24, 36, and 48 h after estrogen stimulation so that **RNA** levels at crit. times throughout cell cycle progression could be monitored. Significant changes include the altered transcript levels of genes implicated in transcription, cellular signaling, and cell cycle checkpoints. At time points during which increased nos. of cells were progressing through S phase, a majority of the genes assocd. with the **DNA** replication fork were also found to be induced. The coexpression of **DNA** replication fork genes by estrogen without the support of serum growth factors indicates an important estrogen regulatory component of the mol. mechanism driving estrogen-induced mitogenesis.
REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 10 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:164299 HCAPLUS
DOCUMENT NUMBER: 131:28397
TITLE: Polymorphism identification and quantitative detection of genomic **DNA** by invasive cleavage of oligonucleotide probes
AUTHOR(S): Lyamichev, Victor; Mast, Andrea L.; Hall, Jeff G.; Prudent, James R.; Kaiser, Michael W.; Takova, Tsetska; Kwiatkowski, Robert W.; Sander, Tamara J.; De Arruda, Monika; Arco, David A.; Neri, Bruce P.; Brow, Marry Ann D.
CORPORATE SOURCE: Third Wave Technologies, Madison, WI, 53719-1256, USA
SOURCE: Nature Biotechnology (1999), 17(3), 292-296
CODEN: NABIF9; ISSN: 1087-0156
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Flap endonucleases (FENs) isolated from archaea are shown to recognize and cleave a structure formed when two overlapping oligonucleotides hybridize to a target **DNA** strand. The downstream oligonucleotide probe is cleaved, and the precise site of cleavage is dependent on the amt. of overlap with the upstream oligonucleotide. We have demonstrated that use of thermostable archaeal FENs allows the reaction to be performed at

temps. that promote probe turnover without the need for temp. cycling. The resulting amplification of the cleavage signal enables the detection of specific **DNA** targets at sub-attomole levels within complex mixts. Moreover, we provide evidence that this cleavage is sufficiently specific to enable discrimination of single-base difference and can differentiate homozygotes from heterozygotes in single-copy genes in genomic **DNA**.

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 11 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:310744 HCAPLUS
DOCUMENT NUMBER: 140:333521
TITLE: Detection of specific nucleic acid sequences using INVADER oligonucleotide-directed cleavage using thermostable derivatives of **DNA** polymerases with thermostable 5'-nuclease activities
INVENTOR(S): Lyamichev, Victor; Neri, Bruce P.; Hall, Jeff; Lukowiak, Andrew
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 393 pp., Cont.-in-part of U.S. Pat. Appl. 2003 152,971.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004072182	A1	20040415	US 2003-356861	20030203
US 5985557	A	19991116	US 1996-756386	19961126
WO 9727214	A1	19970731	WO 1997-US1072	19970122
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
WO 9842873	A1	19981001	WO 1998-US5809	19980324
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6348314	B1	20020219	US 1999-350309	19990709
US 2003152971	A1	20030814	US 2002-290386	20021107

PRIORITY APPLN. INFO.:

US 1996-756386	A3	19961126
WO 1997-US1072	A	19970122
WO 1998-US5809	A	19980324
US 1999-350309	A2	19990709
US 2000-713601	A2	20001115
US 2001-344946P	P	20011107
US 2002-361060P	P	20020227
US 2002-290386	A2	20021107
US 1996-599491	A2	19960124
US 1996-682853	A2	19960712
US 1996-758314	A	19961202
US 1996-759038	A	19961202
US 1997-823516	A	19970324

AB The present invention relates to compns. and methods for the detection and characterization of nucleic acid sequences and variations in nucleic acid sequences by the formation of specific cleavage structures. A 5' nuclease activity from any of a variety of enzymes is used to cleave the target-dependent cleavage structure, with the release of the cleavage product indicating the presence of the target sequences. The cleavage agents and polymerases find use, for example, for the detection and characterization of nucleic acid sequences and variations in nucleic acid sequences. A sample suspected of contg. the target sequence is contacted with oligonucleotides capable of forming an invasive cleavage structure in the present of the target sequence and with an agent for detecting the presence of the invasive cleavage structure. ARRESTOR oligonucleotides

improve sensitivity of multiple sequential invasive cleavage assays and allow use of higher concns. of primary probe without increasing background signal. The detailed description of the invention includes: improved enzymes for the use in INVADER oligonucleotide-directed cleavage reactions comprising **RNA** targets; reaction design for INVADER assay detection of **RNA** targets; kits for performing the **RNA** invader assay; and the INVADER assay for direct detection and measurement of specific **RNA** analytes.

L4 ANSWER 12 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:868661 HCAPLUS
DOCUMENT NUMBER: 136:49292
TITLE: Detection of **RNA** targets using INVADER oligonucleotide-directed cleavage reactions and construction of modified Thermus polymerase enzymes with thermostable 5'-nuclease activities
INVENTOR(S): Allawi, Hatim; Bartholomay, Christian Tor; Chehak, Luanne; Curtis, Michelle L.; Eis, Peggy S.; Hall, Jeff G.; Ip, Hon S.; Kaiser, Michael; Kwiatkowski, Robert W., Jr.; Lukowiak, Andrew A.; Lyamichev, Victor; Ma, Wupo; Olson-munoz, Marilyn C.; Olson, Sarah M.; Schaefer, James J.; Skrzypczynski, Zbigniew; Takova, Tsetska Y.; Vedvik, Kevin L.; Lyamichev, Natalie E.; Neri, Bruce P.
PATENT ASSIGNEE(S): Third Wave Technologies, Inc., USA
SOURCE: PCT Int. Appl., 1266 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001090337	A2	20011129	WO 2001-US17086	20010524
WO 2001090337	A3	20030123		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6759226	B1	20040706	US 2000-577304	20000524
US 2003134349	A1	20030717	US 2001-758282	20010111
US 6635463	B2	20031021		
US 2003104378	A1	20030605	US 2001-864636	20010524
US 2004018489	A1	20040129	US 2001-864426	20010524
JP 2004521606	T2	20040722	JP 2001-587132	20010524

PRIORITY APPLN. INFO.:

US 2000-577304	A	20000524
US 2001-758282	A	20010111
US 2001-864426	A	20010524
US 2001-864636	A	20010524
US 1996-599491	A2	19960124
US 1996-682853	A2	19960712
US 1996-756386	A2	19961126
US 1996-759038	A2	19961202
WO 1997-US1072	W	19970122
US 1997-823516	A2	19970324
US 1999-350309	A2	19990709
US 2000-381212	A2	20000208
WO 2001-US17086	W	20010524

AB The present invention provides novel cleavage agents and polymerases for

the cleavage and modification of nucleic acid. The cleavage agents and polymerases find use, for example, for the detection and characterization of nucleic acid sequences and variations in nucleic acid sequences. In some embodiments, the 5'-nuclease activity of a variety of modified Thermus polymerase enzymes is used to cleave a target-dependent cleavage structure, thereby indicating the presence of specific nucleic acid sequences or specific variations thereof. The term "cleavage structure" refers to a structure that is formed by the interaction of at least one probe oligonucleotide (called the INVADER oligonucleotide) and a target nucleic acid, forming a structure comprising a duplex, the resulting structure being cleavable by a cleavage agent including but not limited to an enzyme. A sample suspected of contg. the target sequence is contacted with oligonucleotides capable of forming an invasive cleavage structure in the present of the target sequence and with an agent for detecting the presence of the invasive cleavage structure. ARRESTOR oligonucleotides improve sensitivity of multiple sequential invasive cleavage assays and allow use of higher concns. of primary probe without increasing background signal. The detailed description of the invention includes: (1) detection of specific nucleic acid sequences using 5'-nucleases in an INVADER-directed cleavage assay; (2) signal enhancement by incorporating the products of an invasive cleavage reaction into a subsequent invasive cleavage reaction; (3) effect of ARRESTOR oligonucleotides on signal and background in sequential invasive cleavage reactions; (4) improved enzymes for the use in INVADER oligonucleotide-directed cleavage reactions comprising **RNA** targets; (5) reaction design for INVADER assay detection of **RNA** targets; (6) kits for performing the **RNA** invader assay; and (7) the INVADER assay for direct detection and measurement of specific **RNA** analytes.

L4 ANSWER 13 OF 294 MEDLINE on STN
ACCESSION NUMBER: 2002622355 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12379459
TITLE: Differential gene expression in gamma-irradiated BALB/3T3 fibroblasts under the influence of 3-aminobenzamide, an inhibitior of parp enzyme.
AUTHOR: Cardoso R S; Espanhol A R; Passos G A S; Sakamoto-Hojo E T
CORPORATE SOURCE: Grupo de Imunogenetica Molecular, Departmento de Genetica, Faculdade de Medicina de Ribeirao Preto, Universidade de Sao Paulo, Ribeirao Preto, SP, Brazil.
SOURCE: Mutation research, (2002 Oct 31) 508 (1-2) 33-40. Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20021017
Last Updated on STN: 20021221
Entered Medline: 20021220

AB 3-Aminobenzamide (3AB) is an inhibitor of poly (ADP-ribose) polymerase (PARP), an enzyme implicated in the maintenance of genomic integrity, which is activated in response to radiation-induced **DNA** strand breaks. cDNA macroarray membranes containing 1536 clones were used to characterize the gene expression profiles displayed by mouse BALB/3T3 fibroblasts (A31 cell line) in response to ionizing irradiation alone or in combination with 3AB. A31 cells in exponential growth were pre-treated with 3AB 4mM 1h before gamma-irradiation (4Gy), remaining in culture during 6h until harvesting time. A31 cells treated with 3AB alone presented a down-regulation in genes involved in protein processing and cell cycle control, while an up-regulation of genes involved in apoptosis and related to **DNA/RNA** synthesis and repair was verified. A31 cells irradiated with 4Gy displayed 41 genes differentially expressed, being detected a down-regulation of genes involved in protein processing and apoptosis, and genes controlling the cell cycle. Concomitantly, another set of genes for protein processing and related to

DNA/RNA synthesis and repair were found to be up-regulated. A positive or negative interaction effect between 3AB and radiation was verified for 29 known genes. While the combined treatment induced a synergistic effect on the expression of LCK proto-oncogene and several genes related to protein synthesis/processing, a negative interaction effect was found for the expression of genes related to cytoskeleton and extracellular matrix assembly (SATB1 and Anexin III), cell cycle control (tyrosine kinase), and genes participating in **DNA/RNA** synthesis and repair (**RNA** helicase, **FLAP endonuclease**-1, **DNA**-3 glycosylase methyladenine, splicing factor SC35 and Soh1). The present data open the possibility to investigate the direct participation of specific genes, or gene products acting in concert in the mechanism underlying the cell response to radiation-induced **DNA** damage under the influence of PARP inhibitor.

L4 ANSWER 14 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:598893 HCAPLUS
TITLE: **DNA** repair and chromatin remodeling: The XPG connection
AUTHOR(S): Tainer, John A.
CORPORATE SOURCE: The Scripps Research Institute, La Jolla, CA, USA
SOURCE: Biochemistry and Cell Biology (2003), 81(3), 261
CODEN: BCBIEQ; ISSN: 0829-8211
PUBLISHER: National Research Council of Canada
DOCUMENT TYPE: Journal; Miscellaneous
LANGUAGE: English

AB We are working to understanding at the mol. level the pathways controlling genetic integrity by characterizing biol. relevant protein-protein and protein-**DNA** interactions for **DNA** repair machinery. Quant. characterization of dynamic conformations and assemblies plus coupled high resoln. X-ray diffraction studies and electron microscopic results aim to integrate **DNA** repair biol. with structure at escalating levels of complexity from domains to multi-protein mol. machines. **DNA** genetic integrity depends upon the structure-specific repair and replication nuclease **Flap EndoNuclease** (FEN-1) and the processivity factor PCNA. FEN-1, which cleaves unpaired over-hanging flaps in double-stranded **DNA** (dsDNA) and removes the terminal priming **RNA** base during **DNA** replication, is necessary for both **DNA** repair and for processing the 50 ends of Okazaki fragments during lagging strand **DNA** synthesis. FEN-1 and PCNA complex structures and mutational results provide a coherent model for substrate recognition and constrain the position of the enzyme when bound to **DNA** and PCNA. These structures and biochem. characterizations furthermore suggest analogous interactions for XPG, which contains sequences homologous to FEN-1. XPG acts in nucleotide excision repair, transcription coupled repair, and base-excision repair. Furthermore, XPG interacts with proteins acting in chromatin remodeling. **DNA** double-strand break repair is a complex process that requires multiple enzymic and structural activities to rejoin or repair the broken ends by one of several repair pathways. These enzymic and structural activities include end detection, end processing, and alignment of **DNA** ends and sister-chromatids. Our recent structural and functional studies show how the Mre11/Rad50 (MR) complex includes both enzymic and structural activities to promote **DNA** end processing and **DNA** linkage in **DNA** recombination and end joining. The nuclease independent functions of the Mre11/Rad50 complex appear to reflect a structural role in facilitating sister chromatid assocn. We have new evidence for a novel structural hook motif that accounts for these nuclease independent functions and explains the function of the striking but least understood feature of the Mre11/Rad50 complex, the 600 .ANG. coiled-coil domain of Rad50. These data help unify architectural and catalytic functions of **DNA** repair complexes in genomic integrity and coordinating repair and chromatin remodeling activities.

L4 ANSWER 15 OF 294 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:384289 HCAPLUS
DOCUMENT NUMBER: 139:95838
TITLE: **DNA** repair and trinucleotide repeat instability
AUTHOR(S): Lahue, Robert S.; Slater, Danielle L.
CORPORATE SOURCE: Eppley Inst. Res. Cancer Allied Diseases, Dep. Pathology Microbiology, Univ. Nebraska Med. Center, Omaha, NE, USA
SOURCE: Frontiers in Bioscience (2003), 8, S653-S665
CODEN: FRBIF6; ISSN: 1093-4715
URL: http://www.bioscience.org/2003/v8/s/1107/pdf.pdf
PUBLISHER: Frontiers in Bioscience
DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English
AB A review. Genes harboring certain trinucleotide repeat (TNR) sequences are at risk for high-frequency mutations that expand or contract the repeat tract. The triplet sequences CNG (where N = any nucleotide) and GAA are known to cause human disease when they expand by more than a few repeats in certain key genes. One o the crucial questions in the field is the mechanism (or, more likely, mechanisms) of triplet repeat expansions and contractions. The available evidence indicates that TNRs can change length as a result of aberrant **DNA** replication in proliferating cells. In addn., TNR instability can arise from gene conversion or by error-prone **DNA** repair whether the cell is dividing or not, since most cell types have recombination and repair activities. The latter of these three sources, **DNA** repair, is the subject of this review because of some recent provocative findings. Two non-mutually exclusive views of **DNA** repair and TNR instability predominant eat this time. One idea is that aberrant **DNA** structure within TNRs blocks repair. Thus even cells with normal repair activities are inhibited from preventing expansions or contractions, due to the local **DNA** structures formed by TNR sequences. A pernicious second model is that **DNA** repair actually contributes to TNR instability. This idea of pro-mutagenic **DNA** repair, although seemingly counterintuitive, has support from a no. of studies. A simple explanation is that repair is triggered either by **DNA** damage in or near the TNR, or perhaps by the aberrant TNR-**DNA** structure itself. Subsequent excision of nucleotides is followed by error-prone repair synthesis. The idea that repair synthesis is a culprit in expansions or contractions ties into the established idea that **DNA** replication through TNRs gives rise to instability. Since **DNA** syntheses also occurs during gene conversion, a common source of TNR instability could well be the errors that arise when **DNA** polymerases attempt to synthesize the problematic triplet repeat sequence.
REFERENCE COUNT: 95 THERE ARE 95 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 11:02:40 ON 24 JUL 2004)

FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, EMBASE, SCISEARCH' ENTERED AT 11:03:32 ON 24 JUL 2004
L1 865 S FLAP ENDONUCLEASE
L2 305 DUP REM L1 (560 DUPLICATES REMOVED)
L3 294 S L2 AND (DNA OR RNA OR POLYNUCLEOTIDE OR DOUBLE FLAP)
L4 294 FOCUS L3 1-

=> s l4 and 1980-1995/py
5 FILES SEARCHED...
L5 10 L4 AND 1980-1995/PY

=> d l5 1-10 ibib ab

L5 ANSWER 1 OF 10 MEDLINE on STN
ACCESSION NUMBER: 96107187 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8530463
TITLE: Calf 5' to 3' exo/endonuclease must slide from a 5' end of the substrate to perform structure-specific cleavage.
AUTHOR: Murante R S; Rust L; Bambara R A
CORPORATE SOURCE: Department of Biochemistry, University of Rochester School of Medicine and Dentistry, New York 14642, USA.
CONTRACT NUMBER: GM24441 (NIGMS)
T32-GM07102 (NIGMS)
SOURCE: Journal of biological chemistry, **(1995 Dec 22)** 270 (51) 30377-83.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199601
ENTRY DATE: Entered STN: 19960220
Last Updated on STN: 19970203
Entered Medline: 19960130

AB Calf 5' to 3' exo/endonuclease, the counterpart of the human FEN-1 and yeast RTH-1 nucleases, performs structure-specific cleavage of both **RNA** and **DNA** and is implicated in Okazaki fragment processing and **DNA** repair. The substrate for endonuclease activity is a primer annealed to a template but with a 5' unannealed tail. The results presented here demonstrate that the nuclease must enter the 5' end of the unannealed tail and then slide to the region of hybridization where the cleavage occurs. The presence of bound protein or a primer at any point on the single-stranded tail prevents cleavage. However, biotinylation of a nucleotide at the 5' end or internal to the tail does not prevent cleavage. The sliding process is bidirectional. If the nuclease slides onto the tail, later binding of a primer to the tail traps the nuclease between the primer binding site and the cleavage site, preventing the nuclease from departing from the 5' end. A model for 5' entry, sliding, and cleavage is presented. The possible role of this unusual mechanism in Okazaki fragment processing, **DNA** repair, and protection of the replication fork from inappropriate endonucleolytic cleavage is presented.

L5 ANSWER 2 OF 10 MEDLINE on STN
ACCESSION NUMBER: 95403394 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7673186
TITLE: Lagging strand **DNA** synthesis at the eukaryotic replication fork involves binding and stimulation of FEN-1 by proliferating cell nuclear antigen.
AUTHOR: Li X; Li J; Harrington J; Lieber M R; Burgers P M
CORPORATE SOURCE: Department of Biochemistry, Washington University School of Medicine, St. Louis, Missouri 63110, USA.
CONTRACT NUMBER: CA51105 (NCI)
GM32431 (NIGMS)
GM43236 (NIGMS)
SOURCE: Journal of biological chemistry, **(1995 Sep 22)** 270 (38) 22109-12.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 19951026
Last Updated on STN: 19951026
Entered Medline: 19951017

AB The 5'-->3'-exonuclease domain of Escherichia coli **DNA** polymerase I is required for the completion of lagging strand **DNA** synthesis, and yet this domain is not present in any of the eukaryotic **DNA** polymerases. Recently, the gene encoding the functional and evolutionary equivalent of this 5'-->3'-exonuclease domain has been identified. It is called FEN-1 in mouse and human cells and RTH1 in Saccharomyces cerevisiae. This 42-kDa enzyme is required for Okazaki fragment processing. Here we report that FEN-1 physically interacts with proliferating cell nuclear antigen (PCNA), the processivity factor for **DNA** polymerases delta and epsilon. Through protein-protein interactions, PCNA focuses FEN-1 on branched **DNA** substrates (flap structures) and on nicked **DNA** substrates, thereby stimulating its activity 10-50-fold but only if PCNA can functionally assemble as a toroidal trimer around the **DNA**. This interaction is important in the physical orchestration of lagging strand synthesis and may have implications for how PCNA stimulates other members of the FEN-1 nuclease family in a broad range of **DNA** metabolic transactions.

L5 ANSWER 3 OF 10 MEDLINE on STN
ACCESSION NUMBER: 95349595 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7623823
TITLE: Mutations in RAD27 define a potential link between G1 cyclins and **DNA** replication.
AUTHOR: Vallen E A; Cross F R
CORPORATE SOURCE: Rockefeller University, New York, New York 10021-6399, USA.
CONTRACT NUMBER: GM7238 (NIGMS)
SOURCE: Molecular and cellular biology, **(1995 Aug)** 15 (8) 4291-302.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950911
Last Updated on STN: 20030204
Entered Medline: 19950829

AB The yeast Saccharomyces cerevisiae has three G1 cyclin (CLN) genes with overlapping functions. To analyze the functions of the various CLN genes, we examined mutations that result in lethality in conjunction with loss of cln1 and cln2. We have isolated alleles of RAD27/ERC11/YKL510, the yeast homolog of the gene encoding **flap endonuclease** 1, FEN-1.cln1 cln2 rad27/erc11 cells arrest in S phase; this cell cycle arrest is suppressed by the expression of CLN1 or CLN2 but not by that of CLN3 or the hyperactive CLN3-2. rad27/erc11 mutants are also defective in **DNA** damage repair, as determined by their increased sensitivity to a **DNA**-damaging agent, increased mitotic recombination rates, and increased spontaneous mutation rates. Unlike the block in cell cycle progression, these phenotypes are not suppressed by CLN1 or CLN2. CLN1 and CLN2 may activate an RAD27/ERC11-independent pathway specific for **DNA** synthesis that CLN3 is incapable of activating. Alternatively, CLN1 and CLN2 may be capable of overriding a checkpoint response which otherwise causes cln1 cln2 rad27/erc11 cells to arrest. These results imply that CLN1 and CLN2 have a role in the regulation of **DNA** replication. Consistent with this, GAL-CLN1 expression in checkpoint-deficient, mec1-1 mutant cells results in both cell death and increased chromosome loss among survivors, suggesting that CLN1 overexpression either activates defective **DNA** replication or leads to insensitivity to **DNA** damage.

L5 ANSWER 4 OF 10 MEDLINE on STN
ACCESSION NUMBER: 95293376 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7774922
TITLE: Sequence of human FEN-1, a structure-specific endonuclease, and chromosomal localization of the gene (FEN1) in mouse

and human.
AUTHOR: Hiraoka L R; Harrington J J; Gerhard D S; Lieber M R; Hsieh C L
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, California 94305-5324, USA.
CONTRACT NUMBER: 5T32CA09302 (NCI)
T32CA09151 (NCI)
SOURCE: Genomics, **(1995 Jan 1)** 25 (1) 220-5.
Journal code: 8800135. ISSN: 0888-7543.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L37374
ENTRY MONTH: 199507
ENTRY DATE: Entered STN: 19950720
Last Updated on STN: 19950720
Entered Medline: 19950707

AB We recently purified and cloned the gene for a **DNA** structure-specific endonuclease, FEN-1, from murine cells. The murine protein recognizes 5' **DNA** flap structures that have been proposed in **DNA** replication, repair, and recombination. Here, we report the sequence of the human FEN1 gene. The translated sequence is identical to peptide sequence obtained from maturation factor-1, which is 1 of the 10 essential proteins for cell-free **DNA** replication. The human protein has the same structure-specific **DNA** endonuclease activity as the murine protein. Two human chromosomal hybridization signals, 11q12 and 1p22.2, were observed by FISH analysis using human genomic clones homologous to the mouse Fen-1 gene. The localization on human 11q12 was confirmed using radiation-reduced hybrids. The mouse Fen-1 gene is assigned to chromosome 19 based on somatic cell hybrids. The significance of these FEN1 gene localizations in human and mouse is discussed.

L5 ANSWER 5 OF 10 MEDLINE on STN
ACCESSION NUMBER: 95181442 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7876218
TITLE: **DNA** structural elements required for FEN-1 binding.
AUTHOR: Harrington J J; Lieber M R
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, California 94305.
SOURCE: Journal of biological chemistry, **(1995 Mar 3)** 270 (9) 4503-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950419
Last Updated on STN: 19950419
Entered Medline: 19950405

AB In eukaryotic cells, a 5'-flap **DNA** endonuclease and a double-stranded **DNA** 5'-exonuclease activity reside within a 42-kDa enzyme called FEN-1 (**flap endonuclease**-1 and 5(five)'-exonuclease). This endo/exonuclease has been shown to be highly homologous to human XP-G, Saccharomyces cerevisiae RAD2, and S. cerevisiae YKL510. Like FEN-1, these related structure-specific nucleases recognize and cleave a branched **DNA** structure called a **DNA** flap and its derivative, called a pseudo Y-structure. To dissect the important structural components of the **DNA** flap structure, we have developed a mobility shift assay. We find that the Fadj strand (located adjacent to the displaced flap strand) is necessary for efficient binding and cleavage of flap structures by FEN-1. When this strand is absent or

when it is present, but recessed from the elbow of the flap strand, binding efficiency drops. Further investigation of the role of the Fadj strand using **double flap** structures reveals that the Fadj strand is necessary to provide a double-stranded template upon which FEN-1 can bind near the elbow of the flap strand. These results provide a basis for understanding how this structure-specific nuclease recognizes a variety of **DNA** substrates.

L5 ANSWER 6 OF 10 MEDLINE on STN
ACCESSION NUMBER: 94178266 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8131753
TITLE: The characterization of a mammalian **DNA** structure-specific endonuclease.
AUTHOR: Harrington J J; Lieber M R
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, CA 94305-5324.
CONTRACT NUMBER: 5T32CA09302 (NCI)
SOURCE: EMBO journal, **(1994 Mar 1)** 13 (5) 1235-46.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L26320
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940428
Last Updated on STN: 19970203
Entered Medline: 19940421

AB The repair of some types of **DNA** double-strand breaks is thought to proceed through **DNA** flap structure intermediates. A **DNA** flap is a bifurcated structure composed of double-stranded **DNA** and a displaced single-strand. To identify **DNA** flap cleaving activities in mammalian nuclear extracts, we created an assay utilizing a synthetic **DNA** flap substrate. This assay has allowed the first purification of a mammalian **DNA** structure-specific nuclease. The enzyme described here, **flap endonuclease**-1 (FEN-1), cleaves **DNA** flap strands that terminate with a 5' single-stranded end. As expected for an enzyme which functions in double-strand break repair flap resolution, FEN-1 cleavage is flap strand-specific and independent of flap strand length. Furthermore, efficient flap cleavage requires the presence of the entire flap structure. Substrates missing one strand are not cleaved by FEN-1. Other branch structures, including Holliday junctions, are also not cleaved by FEN-1. In addition to endonuclease activity, FEN-1 has a 5'-3' exonuclease activity which is specific for double-stranded **DNA**. The endo- and exonuclease activities of FEN-1 are discussed in the context of **DNA** replication, recombination and repair.

L5 ANSWER 7 OF 10 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:550346 HCAPLUS
DOCUMENT NUMBER: 121:150346
TITLE: Functional domains within FEN-1 and RAD2 define a family of structure-specific endonucleases: implications for nucleotide excision repair
AUTHOR(S): Harrington, John J.; Lieber, Michael R.
CORPORATE SOURCE: Lab. Exper. Oncol., Stanford Univ. Sch. of Med., Stanford, CA, 94305-5324, USA
SOURCE: Genes & Development **(1994)**, 8(11), 1344-55
CODEN: GEDEEP; ISSN: 0890-9369
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Structure-specific nucleases catalyze crit. reactions in **DNA** replication, recombination, and repair. Recently, a structure-specific endonuclease, FEN-1, has been purified and shown to cleave **DNA** flap structures. Here, the authors describe the cloning of the murine

FEN-1 gene. The nucleotide sequence of FEN-1 is highly homologous to the Saccharomyces cerevisiae genes YKL510 and RAD2. The authors show that YKL510 and a truncated RAD2 protein are also structure-specific endonucleases. The substrate specificity of the truncated RAD2 protein implicates branched **DNA** structures as important intermediates in nucleotide excision repair. The polarity of these branched **DNA** structures allows the authors to predict the placement of **DNA** scissions by RAD2 and RAD1/RAD10 in this reaction.

L5 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1995:437489 BIOSIS
DOCUMENT NUMBER: PREV199598451789
TITLE: XPG protein has a structure-specific endonuclease activity.
AUTHOR(S): Cloud, Kieran G.; Shen, Binghui; Strniste, Gary F.; Park, Min S. [Reprint author]
CORPORATE SOURCE: Life Sci. Div., LS-1, M888, Los Alamos Natl. Lab., Los Alamos, NM 87545, USA
SOURCE: Mutation Research, (1995) Vol. 347, No. 2, pp. 55-60. CODEN: MUREAV. ISSN: 0027-5107.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Oct 1995
Last Updated on STN: 1 Nov 1995
AB Biochemically active human **DNA** repair protein, xeroderma pigmentosum G (XPG), was overexpressed in insect cells by a recombinant baculovirus. The recombinant baculovirus produced XPG with a mobility of apprx 185 kDa in a denaturing polyacrylamide gel. Indirect immunofluorescence studies demonstrated that the recombinant full-length XPG protein was expressed predominantly as a nuclear protein. The recombinant XPG protein was purified to apparent homogeneity using Q-sepharose, S-300 size exclusion, and Mono Q column chromatography. XPG protein showed a structure-specific **DNA** endonuclease activity, and a preferential affinity to single-stranded **DNA** and **RNA** compared to double-stranded **DNA**.

L5 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1995:326612 BIOSIS
DOCUMENT NUMBER: PREV199598340912
TITLE: Genetic organization of human FEN-1.
AUTHOR(S): Shen, Binghui; Gonzales, Julia A.; Marrone, Babetta L.; Park, Min S.
CORPORATE SOURCE: Life Sciences Div., Los Alamos National Lab., Los Alamos, NM 87545, USA
SOURCE: Journal of Cellular Biochemistry Supplement, (1995) Vol. 0, No. 21A, pp. 303.
Meeting Info.: Keystone Symposium on Repair and Processing of DNA Damage. Taos, New Mexico, USA. March 23-29, 1995. ISSN: 0733-1959.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Aug 1995
Last Updated on STN: 2 Aug 1995

L5 ANSWER 10 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1994:149079 BIOSIS
DOCUMENT NUMBER: PREV199497162079
TITLE: The characterization of a mammalian structure-specific endonuclease.
AUTHOR(S): Harrington, John J.; Lieber, Michael
CORPORATE SOURCE: Dep. Pathol., Stanford Univ. Sch. Med., Stanford, CA 94305, USA
SOURCE: Journal of Cellular Biochemistry Supplement, (1994) Vol. 0, No. 18B, pp. 24.

Meeting Info.: Keystone Symposium on Transposition and Site-Specific Recombination: Mechanism and Biology. Park City, Utah, USA. January 21-28, 1994.
ISSN: 0733-1959.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 Mar 1994
Last Updated on STN: 31 Mar 1994

=> d his

(FILE 'HOME' ENTERED AT 11:02:40 ON 24 JUL 2004)

FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, EMBASE, SCISEARCH' ENTERED AT 11:03:32 ON 24 JUL 2004
L1 865 S FLAP ENDONUCLEASE
L2 305 DUP REM L1 (560 DUPLICATES REMOVED)
L3 294 S L2 AND (DNA OR RNA OR POLYNUCLEOTIDE OR DOUBLE FLAP)
L4 294 FOCUS L3 1-
L5 10 S L4 AND 1980-1995/PY

=> s l2 and 1980-1995/py
5 FILES SEARCHED...
L6 10 L2 AND 1980-1995/PY

=> d l6 10

L6 ANSWER 10 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1994:149079 BIOSIS
DN PREV199497162079
TI The characterization of a mammalian structure-specific endonuclease.
AU Harrington, John J.; Lieber, Michael
CS Dep. Pathol., Stanford Univ. Sch. Med., Stanford, CA 94305, USA
SO Journal of Cellular Biochemistry Supplement, (1994) Vol. 0, No. 18B, pp. 24.
Meeting Info.: Keystone Symposium on Transposition and Site-Specific Recombination: Mechanism and Biology. Park City, Utah, USA. January 21-28, 1994.
ISSN: 0733-1959.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 30 Mar 1994
Last Updated on STN: 31 Mar 1994

=> s double flap nuclease and 1990-1995/py
L7 0 DOUBLE FLAP NUCLEASE AND 1990-1995/PY

=> s flap nuclease and 1990-1995/py
L8 0 FLAP NUCLEASE AND 1990-1995/PY

=> s dna nuclease and 1990-1995/py
L9 71 DNA NUCLEASE AND 1990-1995/PY

=> s dna cleaving activity and 1990-1995/py
L10 145 DNA CLEAVING ACTIVITY AND 1990-1995/PY

=> dup rem l9 l10
PROCESSING COMPLETED FOR L9
PROCESSING COMPLETED FOR L10
L11 104 DUP REM L9 L10 (112 DUPLICATES REMOVED)

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=>  s l11 and 1990-1994/py
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L12          83 L11 AND 1990-1994/PY

=> s l11 and 1990-1993/py
   5 FILES SEARCHED...
L13          65 L11 AND 1990-1993/PY

=> s l13 and endonuclease
L14           3 L13 AND ENDONUCLEASE

=> d l14 1-3 ibib ab

L14  ANSWER 1 OF 3     MEDLINE on STN
ACCESSION NUMBER:    93346840     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 8102169
TITLE:               Gamma-ray-induced transcription and apoptosis-associated
                     loss of 28S rRNA in interphase human lymphocytes.
AUTHOR:              Delic J; Coppey-Moisan M; Magdelenat H
CORPORATE SOURCE:    Institut Curie, Section Medicale et Hospitaliere, Paris,
                     France.
SOURCE:              International journal of radiation biology, (1993
                     Jul) 64 (1) 39-46.
                     Journal code: 8809243. ISSN: 0955-3002.
PUB. COUNTRY:        ENGLAND: United Kingdom
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals; Space Life Sciences
ENTRY MONTH:         199309
ENTRY DATE:          Entered STN: 19930924
                     Last Updated on STN: 19950206
                     Entered Medline: 19930909
AB   Apoptosis, related to a naturally-occurring or programmed cellular death
     process, can be physiologically or exogenously induced. In vertebrate
     cells undergoing apoptosis, initiated by any of these ways, one of the
     numerous biochemical changes is an endogenous endonuclease
     activation that cleaves the chromatin DNA into oligonucleosome-sized
     'ladder' fragments. In the present study we show that in parallel to
     chromatin DNA cleavage, ribosomal RNA is lost in gamma-ray-mediated
     apoptotic human lymphocytes. We demonstrate that 28S rRNA gene
     transcription is induced early (15 min) after irradiation, followed by a
     selective disappearance in apoptotic cells only. The fact that newly
     synthesized rRNA turns over at the same rate in irradiated and untreated
     cell fractions, highly suggests that the observed loss of 28S rRNA in the
     apoptotic cell fraction at the ribosome level is due to degradation
     occurring at a late stage of the apoptotic death process. These results
     suggest that, in addition to first-stage apoptosis-associated rDNA gene
     activation, cellular self-destruction at late stages is associated with
     processes occurring simultaneously at the ribosome level involving an
     endogenous RNase-like activity, and at the chromatin level involving
     DNA-nuclease activity.

L14  ANSWER 2 OF 3     MEDLINE on STN
ACCESSION NUMBER:    93027182     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 1329027
TITLE:               DNA deoxyribophosphodiesterase of Escherichia coli is
                     associated with exonuclease I.
AUTHOR:              Sandigursky M; Franklin W A
CORPORATE SOURCE:    Department of Radiology, Albert Einstein College of
                     Medicine, Bronx, NY 10461.
CONTRACT NUMBER:     R29CA52025 (NCI)
SOURCE:              Nucleic acids research, (1992 Sep 25) 20 (18)
                     4699-703.
                     Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY:        ENGLAND: United Kingdom
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DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199211
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19970203
Entered Medline: 19921104

AB DNA deoxyribophosphodiesterase (dRpase) of E. coli catalyzes the release of deoxyribose-phosphate moieties following the cleavage of DNA at an apurinic/apyrimidinic (AP) site by either an AP **endonuclease** or AP lyase. Exonuclease I is a single-strand specific **DNA nuclease** which affects the expression of recombination and repair pathways in E. coli. We show here that a major dRpase activity in E. coli is associated with the exonuclease I protein. Highly purified exonuclease I isolated from an over-producing stain contains high levels of dRpase activity; it catalyzes the release of deoxyribose-5-phosphate from an AP site incised with **endonuclease** IV of E. coli and the release of 4-hydroxy-2-pentenal-5-phosphate from an AP site incised by the AP lyase activity of **endonuclease** III of E. coli. A strain containing a deletion of the sbcB gene showed little dRpase activity; the activity could be restored by transformation of the strain with a plasmid containing the sbcB gene. The dRpase activity isolated from an overproducing stain was increased 70-fold as compared to a normal sbcB+ strain (AB3027). These results suggest that the dRpase activity may be important in pathways for both DNA repair and recombination.

L14 ANSWER 3 OF 3 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 93:508448 SCISEARCH
THE GENUINE ARTICLE: LT100
TITLE: GAMMA-RAY-INDUCED TRANSCRIPTION AND APOPTOSIS-ASSOCIATED LOSS OF 28S RIBOSOMAL-RNA IN INTERPHASE HUMAN-LYMPHOCYTES
AUTHOR: DELIC J; COPPEYMOISAN M; MAGDELENAT H (Reprint)
CORPORATE SOURCE: INST CURIE, MED & HOSP SECT, RADIOPATHOL LAB, 26 RUE ULM, F-75231 PARIS 05, FRANCE; INSERM, U350, BIOL SECT, F-75231 PARIS 05, FRANCE
COUNTRY OF AUTHOR: FRANCE
SOURCE: INTERNATIONAL JOURNAL OF RADIATION BIOLOGY, (**JUL 1993**) Vol. 64, No. 1, pp. 39-46.
ISSN: 0020-7616.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 36

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Apoptosis, related to a naturally-occurring or programmed cellular death process, can be physiologically or exogenously induced. In vertebrate cells undergoing apoptosis, initiated by any of these ways, one of the numerous biochemical changes is an endogenous **endonuclease** activation that cleaves the chromatin DNA into oligonucleosome-sized 'ladder' fragments. In the present study we show that in parallel to chromatin DNA cleavage, ribosomal RNA is lost in gamma-ray-mediated apoptotic human lymphocytes. We demonstrate that 28S rRNA gene transcription is induced early (15 min) after irradiation, followed by a selective disappearance in apoptotic cells only. The fact that newly synthesized rRNA turns over at the same rate in irradiated and untreated cell fractions, highly suggests that the observed loss of 28S rRNA in the apoptotic cell fraction at the ribosome level is due to degradation occurring at a late stage of the apoptotic death process. These results suggest that, in addition to first-stage apoptosis-associated rDNA gene activation, cellular self-destruction at late stages is associated with processes occurring simultaneously at the ribosome level involving an endogenous RNase-like activity, and at the chromatin level involving **DNA-nuclease** activity.

=> d l13 1-10

L13 ANSWER 1 OF 65 MEDLINE on STN
AN 94064562 MEDLINE
DN PubMed ID: 8244937
TI Genetic and molecular analyses of the C-terminal region of the recE gene from the Rac prophage of Escherichia coli K-12 reveal the recT gene.
AU Clark A J; Sharma V; Brenowitz S; Chu C C; Sandler S; Satin L; Templin A; Berger I; Cohen A
CS Department of Molecular and Cell Biology, Barker/Koshland ASU, University of California, Berkeley 94720.
NC 5371
SO Journal of bacteriology, **(1993 Dec)** 175 (23) 7673-82.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-L23927
EM 199401
ED Entered STN: 19940201
Last Updated on STN: 19950206
Entered Medline: 19940103

L13 ANSWER 2 OF 65 MEDLINE on STN
AN 94053744 MEDLINE
DN PubMed ID: 8235619
TI Crystal structure of neocarzinostatin, an antitumor protein-chromophore complex.
AU Kim K H; Kwon B M; Myers A G; Rees D C
CS Division of Chemistry and Chemical Engineering, California Institute of Technology, Pasadena 91125.
NC CA47148 (NCI)
GM45162 (NIGMS)
SO Science, **(1993 Nov 12)** 262 (5136) 1042-6.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199312
ED Entered STN: 19940117
Last Updated on STN: 19970203
Entered Medline: 19931217

L13 ANSWER 3 OF 65 MEDLINE on STN
AN 94043161 MEDLINE
DN PubMed ID: 7693692
TI Nuclease activities of Moloney murine leukemia virus reverse transcriptase. Mutants with altered substrate specificities.
AU Blain S W; Goff S P
CS Howard Hughes Medical Institute, Columbia University, College of Physicians and Surgeons, New York, New York 10032.
NC CA 30488 (NCI)
SO Journal of biological chemistry, **(1993 Nov 5)** 268 (31) 23585-92.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199311
ED Entered STN: 19940117
Last Updated on STN: 19970203
Entered Medline: 19931129

L13 ANSWER 4 OF 65 MEDLINE on STN
AN 93384585 MEDLINE
DN PubMed ID: 8373405
TI Specific interaction between a novel enediyne chromophore and apoprotein in macromolecular antitumor antibiotic C-1027.
AU Matsumoto T; Okuno Y; Sugiura Y
CS Institute for Chemical Research, Kyoto University, Japan.
SO Biochemical and biophysical research communications, **(1993 Sep 15)** 195 (2) 659-66.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199310
ED Entered STN: 19931029
Last Updated on STN: 19931029
Entered Medline: 19931012

L13 ANSWER 5 OF 65 MEDLINE on STN
AN 93346840 MEDLINE
DN PubMed ID: 8102169
TI Gamma-ray-induced transcription and apoptosis-associated loss of 28S rRNA in interphase human lymphocytes.
AU Delic J; Coppey-Moisan M; Magdelenat H
CS Institut Curie, Section Medicale et Hospitaliere, Paris, France.
SO International journal of radiation biology, **(1993 Jul)** 64 (1) 39-46.
Journal code: 8809243. ISSN: 0955-3002.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 199309
ED Entered STN: 19930924
Last Updated on STN: 19950206
Entered Medline: 19930909

L13 ANSWER 6 OF 65 MEDLINE on STN
AN 93316987 MEDLINE
DN PubMed ID: 7687022
TI The genotoxicity of the waste water discharged from paraquat manufacturing and its pyridyl components.
AU Kuo M L; Lin J K
CS Institute of Toxicology, College of Medicine, National Taiwan University, Taipei.
SO Mutation research, **(1993 Aug)** 300 (3-4) 223-9.
Journal code: 0400763. ISSN: 0027-5107.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199308
ED Entered STN: 19930820
Last Updated on STN: 19980206
Entered Medline: 19930811

L13 ANSWER 7 OF 65 MEDLINE on STN
AN 93194696 MEDLINE
DN PubMed ID: 8449832
TI Analysis of DNA fragmentation in human uterine cervix carcinoma HeLa S3 cells treated with duocarmycins or other antitumor agents by pulse field gel electrophoresis.
AU Okamoto A; Okabe M; Gomi K
CS Pharmaceutical Research Laboratory, Kyowa Hakko Kogyo Co., Ltd.,

Shizuoka-ken.
SO Japanese journal of cancer research : Gann, **(1993 Jan)** 84 (1) 93-8.
Journal code: 8509412. ISSN: 0910-5050.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199304
ED Entered STN: 19930423
Last Updated on STN: 19970203
Entered Medline: 19930409

L13 ANSWER 8 OF 65 MEDLINE on STN
AN 93149094 MEDLINE
DN PubMed ID: 1337138
TI [Specific DNA cleavage by an analog of netropsin containing a copper(II) chelating peptide Gly-Gly-His].
Spetsificheskoe rasshcheplenie DNK analogom netropsina, soderzhashchim khelatiruiushchii ion medi(II) peptid Gly-Gly-His.
AU Grokhovskii S L; Nikolaev V A; Zubarev V E; Surovaia A N; Zhuze A L; Chernov B K; Sidorova N Iu; Zasedatelev A S; Gurskii G V
CS Zhuze AL.
SO Molekuliarnaia biologiia, **(1992 Nov-Dec)** 26 (6) 1274-97.
Journal code: 0105454. ISSN: 0026-8984.
CY RUSSIA: Russian Federation
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 199302
ED Entered STN: 19930312
Last Updated on STN: 19970203
Entered Medline: 19930226

L13 ANSWER 9 OF 65 MEDLINE on STN
AN 93033181 MEDLINE
DN PubMed ID: 1329343
TI Overexpression and purification of enzymatically active recombinant integrase protein of Rous sarcoma virus.
AU Marczinovits I; Molnar J; Soki J; Fodor I
CS Department of Biology, Albert Szent-Gyorgyi Medical University, Szeged, Hungary.
SO Virus genes, **(1992 Aug)** 6 (3) 301-6.
Journal code: 8803967. ISSN: 0920-8569.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199210
ED Entered STN: 19930122
Last Updated on STN: 19970203
Entered Medline: 19921026

L13 ANSWER 10 OF 65 MEDLINE on STN
AN 93027182 MEDLINE
DN PubMed ID: 1329027
TI DNA deoxyribophosphodiesterase of Escherichia coli is associated with exonuclease I.
AU Sandigursky M; Franklin W A
CS Department of Radiology, Albert Einstein College of Medicine, Bronx, NY 10461.
NC R29CA52025 (NCI)
SO Nucleic acids research, **(1992 Sep 25)** 20 (18) 4699-703.
Journal code: 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom

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DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   199211
ED   Entered STN: 19930122
     Last Updated on STN: 19970203
     Entered Medline: 19921104


=> d his

     (FILE 'HOME' ENTERED AT 11:02:40 ON 24 JUL 2004)

     FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, EMBASE, SCISEARCH' ENTERED AT
     11:03:32 ON 24 JUL 2004
L1           865 S FLAP ENDONUCLEASE
L2           305 DUP REM L1 (560 DUPLICATES REMOVED)
L3           294 S L2 AND (DNA OR RNA OR POLYNUCLEOTIDE OR DOUBLE FLAP)
L4           294 FOCUS L3 1-
L5            10 S L4 AND 1980-1995/PY
L6            10 S L2 AND 1980-1995/PY
L7             0 S DOUBLE FLAP NUCLEASE AND 1990-1995/PY
L8             0 S  FLAP NUCLEASE AND 1990-1995/PY
L9            71 S  DNA NUCLEASE AND 1990-1995/PY
L10          145 S  DNA CLEAVING ACTIVITY AND 1990-1995/PY
L11          104 DUP REM L9 L10 (112 DUPLICATES REMOVED)
L12           83 S L11 AND 1990-1994/PY
L13           65 S L11 AND 1990-1993/PY
L14            3 S L13 AND ENDONUCLEASE

=> log y
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                97.27      97.69

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
CA SUBSCRIBER PRICE                               -10.29     -10.29

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FULL ESTIMATED COST                                     0.63       0.63

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FILE 'HCAPLUS' ENTERED AT 10:55:38 ON 24 JUL 2004
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